

Wellen's syndrome: Challenges in diagnosis

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Abstract

Wellen's syndrome is a pre-infarction stage of coronary artery disease characterised by predefined clinical and electrocardiographic (ECG) criteria of a subgroup of patients with myocardial ischaemia. Early recognition and appropriate intervention of this syndrome carry significant diagnostic and prognostic value. We report this unusual syndrome in an elderly man who presented with recurrent angina and characteristic ECG changes as T-waves inversion in the precordial leads, especially in V2–V6 during pain-free periods and ECG obtained during episodes of pain demonstrating upright T-waves with possible elevated ST segments from V1–V4.

Cardiac enzymes were positive and coronary angiography revealed critical stenosis in the proximal left anterior descending artery. It is important to timely identify this condition and intervene appropriately as these patients may develop extensive myocardial infarction that carries a significant morbidity and mortality.

Introduction

Wellen's syndrome was first postulated by de Zwaan et al. in 1982.¹ It is characterised as a disease state in which a patient with angina demonstrates typical electrocardiographic pattern of T-wave changes associated with critical stenosis (>90%) of proximal left anterior descending (LAD) coronary artery.² Discovering Wellen's syndrome is imperative, as these patients are at greater risk of developing anterior wall infarction within few weeks unless intervention is undertaken urgently.³

Case Summary

A 60-year-old elderly man with no previous comorbidities presented to our casualty with complaints of recurrent bouts of retrosternal chest pain, radiating to the left arm, which was mostly present on exertion and subsided on rest. It occurred mostly at morning and sometime in night during sleep. Each episode lasted for 15–25 min. Associated symptoms included profuse diaphoresis, dizziness, shortness of breath and palpitations. Patient had previous episodes of chest pain on exertion, which he overlooked. He had 27-pack year smoking history. He denied any illicit drug use including cocaine. On admission physical examination, patient was afebrile, his pulse rate was 90 beats per minute, blood pressure was 140/100 mmHg, respiratory rate was 18 breaths per minute and saturation on room air (SpO₂) was 97%. Systemic examination was unremarkable.

Basic blood parameters (complete blood cell count, electrolytes, liver and renal functions) and fasting lipid profile were normal. Initial electrocardiogram (ECG) at the time of admission revealed symmetrical and deeply inverted T-waves in precordial leads, especially in V2–V6 during pain-free periods (Figure 1A) and ECG obtained during episodes of pain that occurred after 24 h of admission; demonstrated sharpened upright T-waves with elevated ST segments from V1–V4 (Figure 1B). Cardiac biomarkers CPK-MB was 28 IU/L (normal range: 0–25 IU/L), Troponin T was 0.021 µg/L (normal range: 0.00–0.014 µg/L) and serum blood glucose level was 6.5 mmol/L. Transthoracic echocardiography showed that LAD territory was hypokinetic with moderate left ventricular systolic dysfunction and left ventricular ejection fraction (LVEF) of 40%. The patient was initially managed on anti-platelet, anti-thrombotic (subcutaneous low-molecular weight heparin), nitrates and 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins). A coronary angiogram (CAG) showed critical stenosis (90%) due to a thrombus in the proximal left anterior descending artery (Figure 2).

As the patient had recurrent bouts of retrosternal chest, characteristic precordial T-wave changes and critical stenosis of proximal LAD on CAG, we labeled him as having Wellen's syndrome. He was counseled for revascularisation procedure but he refused to do the same.

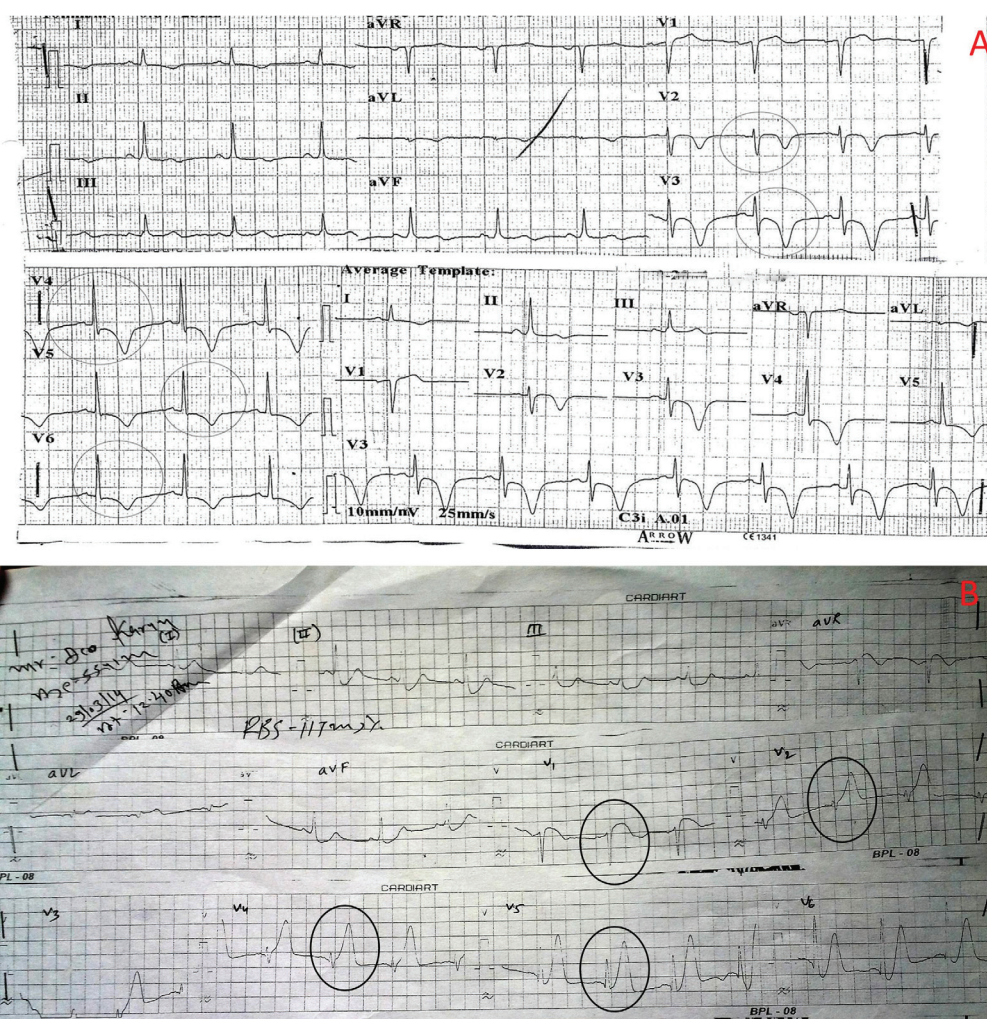


Figure 1. ECG showing deep and symmetrical T wave inversion at V2–V6 precordial leads (A); ST elevation in V1–V4 precordial leads (B)

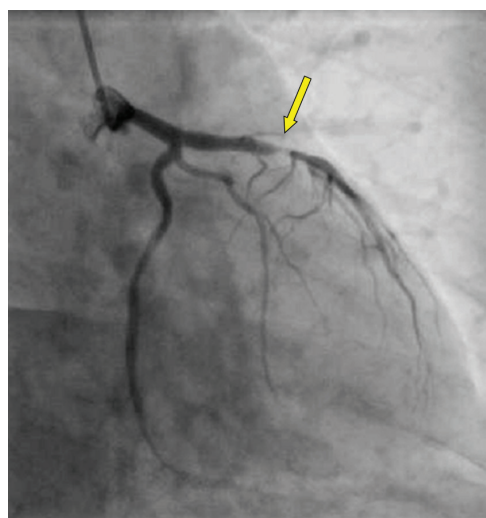


Figure 1. Coronary angiogram showing critical stenosis at the proximal left anterior descending artery

Discussion

Wellens' syndrome has characteristic ECG findings of biphasic T-waves or deep symmetrical T-wave inversions in the precordial leads (leads V1–V4). This ECG finding usually occurs during a pain-free period and is highly suggestive of critical proximal LAD coronary artery stenosis.⁴ The patient also had similar T-wave inversion during pain-free period and had ST elevation on ECG during pain.

Patients often present with angina and found to have specific precordial T-wave with high-degree stenosis of the proximal LAD coronary artery.¹ Two variations of Wellens' syndrome T-wave have been notified. Type A is most common and occurs in 75% of cases. It is characterized by deeply inverted T-waves in V2 and V3. Type B occurs in 25% of cases and is illustrated by biphasic T-waves in V2 and V3.⁵

Diagnosis of this syndrome is difficult, as most of the patients do not have chest pain during the visit, have normal or minimally elevated cardiac enzymes and non-specific ECG findings. Therefore, it is important to compare serial ECGs, as this might provide clues to the diagnosis.⁶

The patient in this study had retrosternal radiating chest pain with slightly raised cardiac enzymes with ECG finding suggesting non ST elevation myocardial infarction (NSTEMI). NSTEMI in the patient was likely due to vulnerable atherosclerotic plaque in the proximal

LAD coronary artery. The angiographic pattern of the disease was typical of the underlying atherosclerosis. Patient had cardiovascular risk factors, such as smoking and hypertension that we believe synergistically accelerated atherosclerosis.

Conflict of interest

None.

Source of funding

Nil.

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